

**SURGICAL TREATMENT OF PERIPHERAL FACIAL PARALYSIS***HAN Weiju*

The facial nerve passes through the temporal bone and is the longest nerve that travels in a bony canal with a complex course and high susceptibility to injury. When facial nerve becomes swollen from insults such as trauma, inflammation, tumor or iatrogenic injury, its distal portion can suffer ischemia and degeneration from increased pressure as a result of lack of expansion room in a rigid bony canal, potentially leading to severe nerve damage and even necrosis and fibrosis<sup>[1]</sup>. Facial paralysis can produce significant impact on the patient both psychologically and in daily life, and should be treated in a timely manner. Management of peripheral facial paralysis may involve otolaryngologists, ophthalmologists, facial plastic surgeons and psychologists<sup>[2, 3]</sup>. During the acute stage, the treatment is primarily focused on eliminating the underlying cause. For example, for facial paralysis secondary to acute otitis media, treatment is aimed at controlling infection in the middle ear. In Bell's palsy, oral steroids at early time often lead to good outcomes<sup>[4-7]</sup>. When caused by compression from trauma and fracture, early surgical intervention should be the treatment of choice<sup>[8]</sup>.

Surgical treatments for facial paralysis include decompression, nerve repair and late stages plastic surgeries<sup>[9, 10]</sup>. PLA General Hospital Department of Otolaryngology Head and Neck Surgery is among the first in China to provide surgical treatments for peripheral facial paralysis. Over the years, thousands of cases have been treated by generations of otologic surgeons in our department. Common etiologies in these cases include Bell's palsy, trauma, iatrogenic injury, middle ear infection, temporal bone cholesteatoma and facial nerve tumors. Among methodologies for surgical intervention are facial nerve decompression via mastoid-epitympanum, middle fossa or combined mastoid-middle fossa approaches, end-to-end facial nerve anastomosis, translocation anastomosis, great auricular nerve grafting and facial-hypoglossal anastomosis,

with good results in most cases<sup>[11-14]</sup>.

**Facial nerve decompression**

Because the facial nerve passes through the bony facial nerve canal, swelling of the nerve as a result of inflammatory or non-inflammatory edema from various etiologies can lead to increased pressure within the canal. Sustained high pressure can block axoplasm flow and growth of regenerated nerve toward facial musculature it supplies. For this reason, in peripheral facial paralysis that is unlikely to recover on its own, prompt decompression is critical. Decompression is used in peripheral facial paralysis caused by a broad range of etiologies, on the premises that the continuum of the nerve remains intact and that nerve injury is less than 1/3 of the nerve main trunk. Decompression requires opening the bony canal and nerve sheath to release pressure and reduce compression on nerve fibers. The goal of decompression is to improve blood circulation and minimize damage to distal nerve fibers.

Understanding of indications and timing for facial nerve decompression has gone through a historical evolution process, along with increasing understanding of pathogenesis of peripheral facial paralysis. Even today, there are no universally accepted criteria and further proof by clinical trials is still needed existing proposals. In the 1930s, Balance et al conducted decompression of facial nerve at the stylomastoid foramen and within the mastoid segment up to 1 cm above the foramen in patients with Bell's palsy whose facial function had not recovered over 3 months. In the 1940s and 1950s, the indication for decompression was modified to cover patients whose facial function recovery was less than ideal over 2 months, and the range of decompression was increased to cover the entire mastoid, pyramidal and tympanic segments of the facial nerve. In the 1960s, the indication

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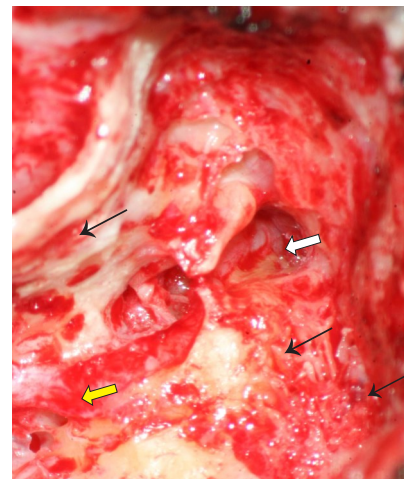
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was yet again modified to include cases of facial paralysis within 2 weeks of onset and with severe facial nerve damage, and full course facial nerve decompression started. In the 1970s to 1980s, earliest time for surgery was shortened to 10-15 days within onset, and decompression of the labyrinthine and internal auditory canal segments of facial nerve via the middle fossa approach was developed. All these procedures were mainly for Bell's palsy<sup>[15, 16]</sup>. With intraoperative facial nerve monitoring, Fisch found that the site of blockage in Bell's palsy was at the entrance of facial nerve canal near the end of the internal auditory canal-the narrowest point of the canal (diameter = 0.7 mm). He believed that edema of the facial nerve in this area was the main cause of facial paralysis. He also considered decompression via a supra-labyrinthine approach (e.g. the middle fossa approach) as an effective method in treating Bell's palsy. In the first 2-3 weeks of facial paralysis, 90% or greater of degeneration on ENoG was recognized as indication for decompression surgery<sup>[17]</sup>. The results in a multi-center prospective study by Gantz et al further confirmed Fisch's point, i.e. facial nerve decompression within the first 2 weeks when indicated yields significant recovery of nerve function and improvement of facial function and swelling of the nerve at the entrance to the facial nerve canal is the key cause of Bell's palsy<sup>[16]</sup>. WANG Zhengmin et al believe that 90-94% degeneration within the first 3 weeks is an indication for emergency decompression. Even if the course is longer than 3 weeks and degeneration on ENoG is 95-100%, depression may result in improved facial function in most cases<sup>[10]</sup>.

In Bell' palsy, when degeneration is greater than 90% within the first 2 weeks, decompression can improve clinical prognosis and facial function. It is worth noting that ENoG reflects the extent of distal nerve degeneration only when wallerian degeneration starts within the first 4-14 days. Decompression surgery within the first 2 weeks following complete facial paralysis can improve the overall facial recovery<sup>[15, 18]</sup>. A multi-center prospective clinical study on ENoG guided facial nerve decompression showed that facial function achieved House-Brackmann I or II grade in 7 months in all cases when degeneration was less than 90% in the first 2 weeks, but only II or III grade if degeneration in the first 2 weeks was greater than 90% with no motor unit potentials, although facial function was restored to near normal after decompression of the nerve proximal to the geniculate ganglion. For best possible treatment results, decompression is recommended in patients with complete facial paralysis within the first 2 weeks<sup>[16, 19]</sup>. If hearing is normal, decompression of the labyrinthine and geniculate segments via the middle fossa approach is recommended, whereas transabyrinthine approach can be

considered when there is no useful hearing<sup>[20]</sup>. However, some reports indicate good results in cases where facial paralysis has been present for 3-6 months or even longer.

In case of facial paralysis following head trauma, we recommend exploration and decompression of the facial nerve at the earliest time possible if there is immediate complete facial paralysis and patient is stable. Fractured bone debris and blood clot should be removed and nerve sheath incised for decompression of the facial nerve and prevention of secondary nerve damage from edema and elevated pressure. For those with delayed or incomplete facial paralysis, criteria for Bell's palsy may be applied. Following initiation of steroid treatment, ENoG test can be performed. If ENoG shows degeneration greater than 90% within 4-14 days, exploration and decompression of the facial nerve should be considered<sup>[21]</sup>. Based on the orientation of fracture lines in relation to the petrous bone, temporal bone fracture can be classified as longitudinal and transverse types. Longitudinal fracture can cause ear drum perforation, ossicular chain damage, conductive hearing loss, ear canal bleeding and facial nerve damage at the horizontal and/or geniculate segments. Decompression in such cases can be completed via mastoid-epitympanic approach (Figure 1). Transverse fracture line is perpendicular to the petrous bone across the otic cyst and result in inner ear damage, sensorineural hearing loss, vertigo and damage to the facial nerve at the labyrinthine segment. For these cases, middle fossa or translabyrinthine (with severe sensorineural hearing loss) or middle fossa-mastoid approach can be used for local or full course decompression.



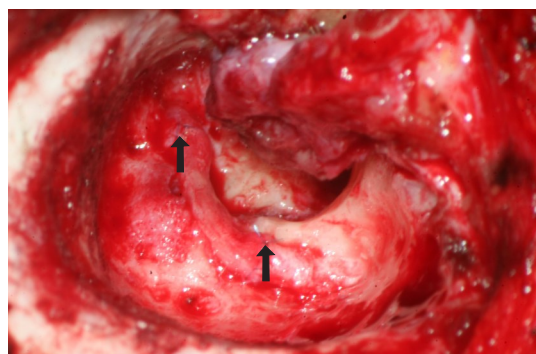
**Figure 1.** Facial nerve decompression via mastoid-epitympanum approach in a patient with longitudinal temporal bone fracture and complete facial paralysis. The facial nerve is decompressed from the vertical segment to the geniculate ganglion and the ossicular chain remains intact (← : fracture line; ← : geniculate ganglion; ← : vertical segment of facial nerve)

## Facial nerve repair

Facial nerve repair involves end-to-end anastomosis and nerve grafting. When exploring and decompressing the facial nerve, if the nerve is completely or nearly completely transected and the stumps can be located, end-to-end anastomosis is the treatment of choice. End-to-end anastomosis is frequently performed following resection of tumors of the facial nerve or temporal bone or in case of iatrogenic facial nerve injury, although it can be used in some cases of temporal bone fracture and other open injuries. In case of complete transection with no loss of the nerve (often seen in accidental injury during surgeries and open trauma), direct tensionless end-to-end anastomosis is possible. In case of severe facial nerve damage, if loss of nerve is less than 3 mm, end-to-end anastomosis can be completed after nerve translocation, i.e. mobilizing the nerve from the bony canal after opening the canal from the geniculate ganglion to the stylomastoid foramen, removing damaged nerve section and stitching the nerve sheath (avoiding damaging the perineurium) using 8-0 absorbable sutures<sup>[22]</sup>.

If direct or translocation end-to-end anastomosis is not possible (or if it creates excessive tension on the nerve), nerve grafting will be needed. Because regenerated nerve has to pass through two anastomotic junctions in nerve grafting, treatment results are slightly inferior to direct end-to-end anastomosis but still superior to facial-hypoglossal anastomosis<sup>[23]</sup>. In resecting facial nerve tumors, due to the tight relation between the tumor and nerve, it is often difficult to track and separate the nerve and facial nerve can be compromised in the process of removing the tumor. Nerve grafting is often needed in such cases<sup>[12]</sup>. Facial nerve injury from mastoid surgeries is often severe with significant nerve loss and requires nerve grafting (Figure 2). The great auricular or peroneal nerve can be used as the graft. The great auricular nerve is nearby and easily available by extending auricular incisions, but may have variable sizes. As long as 10 cm of graft may be available from the great auricular nerve, usually plenty for facial nerve repair. When longer graft is needed, the peroneal nerve may be the choice, which has consistent sizes similar to that of the facial nerve. As long as 20 cm or longer can be available, although a separate surgical field will be needed and sensation in the leg will be compromised<sup>[24]</sup>, sometimes resulting in hercules tendon rupture during vigorous activities.

When facial nerve is accidentally damaged during middle ear or mastoid surgeries, immediate treatment is needed, including end-to-end anastomosis and nerve grafting. If facial paralysis is identified after surgery, treatment will be dictated by the timing and severity of



**Figure 2.** Repair of facial nerve horizontal and pyramidal segments with great auricular nerve graft ( ← :arrow heads)

paralysis. Delayed facial paralysis often recovers on its own. For immediately present facial paralysis, effects of local anesthesia and tight dressing should be ruled out first. If persistent paralysis, the surgical course must be reviewed for possible intraoperative damage to the facial nerve. If confident of intact facial nerve integrity, continuing observation is reasonable and the nerve may be explored when ENoG indicates more than 90% degeneration. However, if intraoperative facial nerve damage is suspected or questionable through surgery course review, revision and nerve exploration should be conducted as soon as possible, with the patient's or guardian's informed consent. Severe edema or granulation from prolonged delay may increase difficulties in identifying the nerve. The surgeon should be ready to perform anastomosis or grafting procedures during revision<sup>[13, 25-27]</sup>.

## Facial-hypoglossal anastomosis

In facial nerve damage in which the proximal stump is not available for anastomosis or grafting, alternative nerves can be considered to restore neural supply to facial musculature, with the hypoglossal nerve being the first choice. Facial-hypoglossal anastomosis is a type of dynamic plastic or nerve transplantation procedure. It provides improved stationary facial symmetry and tone and voluntary facial movement in some cases with rehabilitation training. The hypoglossal nerve is usually selected because it may be synergic with the facial nerve from functional and developmental perspectives. At cortex level, areas representing facial and tongue motor functions are closely located. Both end-to-end and end-to-side anastomosis techniques are used in facial-hypoglossal anastomosis. While end-to-end anastomosis provides more facial animation, it requires sacrifice of the entire hypoglossal nerve, leading to hemi-tongue atrophy and sometimes articulation difficulties. In contrast, end-to-side



technique may provide better preservation of tongue function.

Regardless of the etiology of facial nerve injury, as long as the distal stump can be located with bilateral normal hypoglossal nerves and no facial muscle fibrosis, facial-hypoglossal anastomosis can be performed to improve facial function. For patients with complete facial paralysis, facial-hypoglossal anastomosis should be completed within 1 year. When longer than 1 year, facial-hypoglossal anastomosis may still be considered if EMG continues to show fibrillation potentials, although the patient should be informed of the possible less than ideal prognosis. For professional singers or speech givers who cannot tolerate compromised tongue function, facial-accessory nerve anastomosis can be considered, although shoulder asymmetry and facial synkinesis upon lifting shoulder are possible complications. Our group has used facial-hypoglossal anastomosis primarily in cases of facial nerve tumor in the internal auditory canal where the proximal stump cannot be located, facial nerve injury during resection of large acoustic neuroma, temporal bone tumors or petrous cholesteatoma compromising the facial nerve where proximal stump cannot be located, and facial nerve injury of other etiologies where facial nerve end-to-end anastomosis or grafting repair are not possible<sup>[11, 12]</sup>. Facial-hypoglossal anastomosis can improve facial function to H-B III or IV and reduce facial musculature atrophy, although frontal crease symmetry is often not restored.

For late stage facial paralysis, there are dynamic and non-dynamic therapies. Dynamic therapies include the facial-hypoglossal/accessory anastomosis (as described above), as well as nerve grafting and grafting with pediculated neuromuscular flap for restoration of facial animation. Cutaneous or fascia suspension techniques are among non-dynamic therapies used to improve aesthetic experiences. These are palliative therapies for cases in which facial nerve repair is no longer possible.

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